What is of greater concern is the way in which your correspondents have sought to obscure the message of our paper. This message is that patients in our study were unhappy about charges which had inflated at 24% per annum. No amount of analysis of the total fee, of GST or of general practitioner incomes will change beese facts. Nor will they be altered by claims that patients have ho right to determine what they deem to be a fair and reasonable cost for medical care.

Further, some of your correspondents imply that the purpose of our paper was to criticise general practice and to bring general practitioners into disrepute. This is not so, as will be seen from our previous article (i) on this topic. The purpose of the present article was not to denigrate the efforts of general practitioners but rather to bring to attention a current public perception of the cost of their services. We would hope that this evidence will contribute positively to policy attempts to devise a fee structure which ensures that family doctors receive a fair return for their efforts while at the same time protecting patients from the current inflation in patient charges which has largely been caused by the way in which the relationships between the botal fee and GMS are set.

D M Fergusson.. L J Horwood. F T Shannon.

Christchurch Child Development Study. Christchurch Hospital. Christchurch.

Ferguseon DM, Beautrais AL, Shannon FT, Maternal saturfaction with primary health care. NZ MedilJ 1961, 94: 2914.

## Deaths from lung cancer and ischaemic heart disease due to passive smoking in New Zealand

Kawachi. Pearce and Jackson (1) estimate that passive smoking causes 273 deaths per year in New Zealand, 30 from lung cancer and 243 from ischaemic heart disease (IHD). Some 65% of these deaths are attributed to workplace exposure, the rest to spousal smoking. These estimates are scientifically unjustified. Too much weight is given to fragile epidemiological data, major sources of bias being totally underestimated. Too little weight is given to evidence that nonsmokers have very low exposure to tobacco smoke constituents.

The evidence that passive smoking increases risk of IHD is very unconvincing. The authors [1] cite a meta-analysis by Wells [2] for their estimate of risk in relation to spouse smoking. This is based on 7 studies, many of which involve unacceptably small numbers of cases, eg, as low as two deaths in women married to never smoking husbands (3). The two studies with adequate numbers are both open to question.

One of these is the Japanese prospective study (4.5). Wells cited results from 17 years follow up (5) which claimed a significant trend in IHD in relation to spouse smoking, but failed to mention that this finding significantly (p < 0.001) conflicted with an earlier report, based on:14 years follow up which claimed no association whatsoever!

The other is the Maryland prospective study spenich reported 34% and 24% increases in IHD in men and women in relation to spouse smoking. This study has many features that are notaworthy. It made no attempt to follow up people moving outside Washington County, thus missing large numbers of deaths. It found no dose response relationship. It failed to collect data on a whole range of possibly relevant confounding factors. Those it did adjust for (age, marital status, years of school, quality. of housing) had an enormous effect on relative risk, changing estimates from 1.17 to 1.31 in men and from 0.66 to 1.24 in women. emphasising the fragility of the results.

The evidence relating passive smoking to lung cancer is more extensive than for IHD; being based on 27 published studies, not 13 as Kawachi et al state! While there is an association of spouse smoking to lung cancer risk that cannot plausibly be explained by publication bias, it cannot be reliably inferred this results from a causal effect of passive smoking. In the first place, exposure of nonsmokers to smoke constituents is very low. Thus typical nonsmokers retain only about 0.01-0.02% of the amount of smoking related particulates retained by a smoker [7]. Furthermore, there are various sources of persistent bias in the epidemiology, a major one caused by misclassification of a

proportion of smokers as nonsmokers. As argued at length elsewhere [8-12]; this bias can produce an artefactual association of a similar magnitude to the association claimed by Kawachi et all[1] to be due to passive smoking. Wells [2] correction for this bias was totally inadequate, failing to allow for the possibility of misclassified current typical regular smokers, whereas a recent summary of data from large studies shows an average rate of about 4% [11].

Although there is virtually no epidemiological data on risk in relation to workplace exposure to passive smoking. Kawachi et al (I) present estimates based on unjustified extrapolations from the spouse smoking estimates, which are themselves hopelessly biased.

The authors present numbers of deaths with ranges, so giving the uninformed reader a spurious idea of accuracy. When one considers no major authority has yet concluded passive smoking causes IHD; it is difficult to see what useful meaning one can attach to the cited lower limits of 39 IHD deaths for spousal smoking and 62 for workplace exposure.

Peter N' Lee (Mr): PN Lee Statistics and in a material at the Computing Ltd. Cedar Road. Chateured by 2000 or 312 Sutton. dw. The 17 U.S. Dice Surrey SM2 5DA, UK.

- Kawachi I Pearce NE, Jackson RT. Deaths from lung cancer and unchasence heart diseases due to passive smoking in New Zealand. NZ Med J 1969: 102: 337-40.
   Wells AJ. An estimate of adult mortality in the United States from passive smoking Environment International 1965. 18: 249-85.
   Garland C. Barrett-Connor E. Suarez L. et al. Effects of passive smoking on inchesing the control of t
- 645-50
  Hirsyama T. Non-smoking wives of beavy smokers have a higher risk of hing centure study from Japas. Br Med J 1961, 282, 183-5.
  Hirsyama T. Ling cancer in Japan, effects of nutrition and passive smoking. In: Missell M. Cortes P.; eds. Ling cancer causes and prevention. New York, Verlag Chemie International, 1964; 175-95.
  Helming KJ, Sandler DP, Comstock GW, Chee E, Heart disease mortality in nonemokers.

- rammag rul: bancier DP: Comstock GW, Chee E. Heart disease mortality in nonemokers living with amokers. Am J Epidemiol 1988, 127::915-22.

  Arusdel A. Starling T. Weinkam J. Never smoker long cancer risks from exposure to persuculate tobacco smoke. Environment: International 1987, 13, 409-26.

  Les PN. Passivs smoking and long cancer. Assomation a result of bias? Human Toxicol 1987, 6: 517-24.
- Lee PN. Misclinenfication of smoking habits and passive smoking. A review of the evidence. International Archives of Occupational and Health Supplement. Headelberg. Springer-Verlag. 1988.

  Lee PN. An International Archives Les PN. MisciaenScation of smok
- Lee PN: An alternative explanation for the increased risk of lung cancer is non-escalar married to smokers. In: Perry R. Kirk PW: eds. Indoor and embient ar quality. London.
- Setpar: 1985-149-36. Les PN. Passive smoking: Fact or School\* Paper presented at:Conference on Pres and Puture of Indior Air Quality held in Brusenis on February 14-16, 1985. Les PN. Problems in interpreting epidemiological data. Paper presented at Comfere on Assessment of Inhalation Hasards held in Hazover on February 19-24, 1989.

## Pailed vasectomy

A recent ACC appeal case was published in the Otago Daily Times and I Yound it very disturbing (see Medicologal p 453). This couple was awarded compensation after the alleged failure of a vasectomy performed at Oamaru Hospital in 1979. After this operation it took nine manths before the sperm count was zero. Nearly five years later the appellant's wife fall pregnant. These happenings can be easily explained in that the vasectomy was performed correctly but the sperm count took a long time to reach zero because the patient was slow to ejaculate all the sperm from his body. This is quite often seen. The pregnancy resulted from recannalisation of the vas deferens and can occur once in about every 500 vasectomies. Yet despite the above explanations, some other surgeon has stated that it is standard medical practice to recommend a repeat vasectomy after three or at the most four positive sperm tests after a vasectomy. Judge Middleton has accepted this surgeons evidence as gospel and this persuaded the judge to allow the claim. If the facts of the case are as I read them in the Otago Daily Times then there has been a clear miscarriage of justics.

Compensation has been wrongly awarded and a doctor wrongly accused of negligence. This case may set a false precedent. The Accident Compensation Corporation abould not be allowed to accept this appeal decision and this case should go to a higher court.

J K Walton

Department of Urdiogy. Dunedin Hospital